THROMBOPROPHYLAXIS IN GENERAL SURGERY

(with a Synopsis of Venous Thromboembolism)

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Citation: *Mbah N. Thromboprophylaxis in General Surgery (with a synopsis of Venous Thromboebolism). Orient Journal of Surgical Sciences. March 2020; 1 (1): 1 - 9*

ABSTRACT

Venous thromboprophylaxis aims at reducing avoidable morbidity, death and chronic ill health from hospital associated venous thromboembolism (VTE). Reports indicate little awareness of VTE and inadequate provision of primary thromboprophylaxis on surgical patients managed in most hospitals in Africa. Clinical practice guidelines (CPG) regarding VTE risk assessment, risk categorization and preventive methods using mechanical means and pharmacological agents for the patients at risk are virtually non-existent in most of our health institutions. Compliance with such guidelines where available is poor due to lack of enforcement.

This article is an overview of VTE and the provision of thromboprophylaxis for hospitalized general surgery patients.

It is strongly recommended that a hospital-based thromboprophylaxis guideline be formulated and enforced at each surgical service in our health community in order to optimise patients outcome .

Key words: Thromboprophylaxis; VTE; Clinical Practice Guidelines; General Surgery.

INTRODUCTION

The goal of every clinician is to deliver high quality evidence-based care to their patients. One of the factors which subvert the optimal post-operative outcome in the surgical patient is the development of venous thromboembolism (VTE) either as deep vein thrombosis (DVT) or its more fatal complication, the pulmonary embolism (PE).

Venous thromboembolism refers to blood clot formation within the venous circulation. It could manifest as superficial thrombophlebitis, deep vein thrombosis (DVT) or as pulmonary embolism (PE).

Deep vein thrombosis (DVT) is blood clot within the deep veins of the calf, thigh, pelvis or less commonly the arm or neck.

Pulmonary embolism develops when a clot detaches from a DVT and migrates to occlude the blood vessels of the lung.

Clinically, the practice of primary thromboprophylaxis, which are measures taken to prevent venous thrombosis, is directed at both DVT and PE but not superficial thrombophlebitis which is a less severe form of venous thrombosis within the superficial veins.

The prevention of VTE is the number one strategy to improve patient care according to the United States Agency for Health Care Research and Quality.[1]

The scope of this article is VTE and thromboprophylaxis in general surgery patients.

EPIDEMIOLOGY

Venous thromboembolism (VTE) is a very common public health problem. It is an important preventable cause for morbidity and mortality among patients who undergo general surgery.[2]

Several studies have identified PE as the most common preventable cause of hospital deaths.[3, 4, 5]

It's estimated that 1-2 of every 1,000 Americans are diagnosed with VTE every year.

Furthermore, 60,000 - 100,000 Americans die of venous thrombosis annually. More Americans die from VTE each year than from acquired immune deficiency syndrome (AIDS) and breast cancer combined.[6]

In Europe, the total annual burden of VTE across the 25 member states of the European Union (before Brexit), with a population of 454 million, was estimated to be 640,000 symptomatic cases of DVT and 383,000 PE. VTE –related deaths were estimated at 480,000 annually. In the United Kingdom (UK) with 60 million inhabitants, an estimated 60,000 people die from preventable hospital acquired VTE every year.[7]

With regards to the surgical patient, VTE is one of the more common complications seen in patients following surgery, cancer, trauma or prolonged immobilization.[8]

Deep vein thrombosis (DVT) has been estimated to occur in up to 40% of postoperative patients without thromboprophylaxis.[9] The morbidity and mortality associated with thromboembolic events is high, with 28-day fatality rates reported as 9% for DVT and 15% for pulmonary embolism (PE).[10, 11]

Although the data on this disease in Africa is scanty, a meta-analysis of studies reported by Danwang et al showed a post-operative DVT prevalence of 2.4% - 9.6% and a case-fatality rate of 60% from pulmonary embolism (PE) among surgical patients across various centres in the African continent.[12] They also observed that at least one

quarter of patients at risk for VTE in Africa did not receive thromboprophylaxis. Their findings indicate little awareness of VTE in Africa which consequently reflects the inadequate attention which is paid to thromboprophylaxis practice across the primary, secondary and tertiary health facilities in this environment. The prevention of VTE in the surgical patient, as for the prevention of any other disease condition, is predicated on adequate understanding of its background, the synopsis of which would be presented in this article.

AETIOLOGY OF VTE

Deep vein thrombosis (DVT) develops primarily due to any, or a combination, of the 3 components of Virchow's triad, namely injury to the endothelial lining of the vein wall, alteration of blood flow and hypercoagulability of blood.[13]

Pulmonary embolism develops from the detached and migrated clot from DVT mainly of the pelvis and lower limb.

RISK FACTORS

1. Age. The incidence of venous thrombosis is greater in patients over 40 years of age and almost doubles with each subsequent decade of life thereafter.[14] Venous thromboembolism was previously considered to be rare in children below 16 years of age. However, the recognition and diagnosis of VTE in hospitalised children has increased due to greater VTE awareness, higher level of suspicion in particular clinical contexts, increased availability of imaging techniques, the longer survival of children with previously fatal or chronic diseases that can predispose to thrombosis, and to the more frequent use of central venous lines.[15, 16, 17, 18] Indeed some have reported a 3- to 10-fold rise in VTE incidence among hospitalised children since the beginning of the 21st century.[19, 20]

2. Previous history of VTE. The recurrence of venous thrombosis is estimated at 5 -7% annually after the first episode.[21, 22, 23]

3. Drugs. Both legal and illegal drugs are associated with increased risk for VTE. These include, but are not limited to oestrogen-containing contraceptive pills, tamoxifen, chemotherapeutic cytotoxic drugs, hormone replacement pills, antipsychotic drugs, radiologic contrast media and the paradoxical throemboembolic complication associated with heparin, otherwise known as heparin induced thrombocytopaenia (HIT).[24, 25]

Others are non steroidal anti-inflammatory drugs (NSAIDs), marijuana, cortisone, gonadotropins, sirolimus, herbal concoctions, anti-vascular endothelial growth factors (VEGF), sildenafil, cocaine and ephedrine.[26, 27, 28, 29, 30, 31]

4. Venous stasis from immobility. Predisposing conditions include minor forms of immobility such as following minor surgery or injury.[32] Major surgery lasting > 30minutes, prolonged recumbency during the post-operative period, long distance travel by car, train or air, obesity, sedentary life style, paralysis from cardiovascular accidents (stroke) or spinal cord injury, major illnesses eg congestive cardiac failure, renal failure, plaster casts on the legs, all have been linked to venous thrombosis risk.[21, 33, 34, 35]

5. Vascular injury causes VTE. This could follow civilian or military injuries, surgery or vascular catheter placement particularly the central intravenous (IV) canulation.[36]

6. Hypercoagulable States from dehydration, malignancy, inflammatory bowel disease (IBD), antiphospholipid antibodies as seen in systemic lupus erythematosus (SLE), myeloproliferative disorders (such as polycythaemia rubra vera and essential thrombocythaemia), pregnancy and puerperium are predisposing factors for VTE.[37, 38]

7. **Hereditary Disorders** associated with hypercoagulable state. The inherited deficiencies of physiological anticoagulants such as protein C deficiency, protein deficiency, antithrombin (AT) III deficiency and activated protein C resistance (APC) could lead to VTE.[39] Other inherited thrombophilic conditions are factor V Leiden (FVL) disorder and prothrombin mutation G20210A.[15, 37, 38]

CLINICAL MANIFESTATION OF VTE

A. DVT – This condition may be silent in majority of cases.[38] Symptomatic DVT manifests as unilateral or bilateral leg (or sometimes) arm swelling, pain in the affected limb, change in the overlying skin colour and warmth at the area of swelling. Patients with post thrombotic syndrome, caused by damage to venous valves with development of chronic venous insufficiency, present with chronic limb swelling, pain, tenderness, dark discolouration of overlying skin, scaling, itching and ulceration, most of which constitute lipodermatosclerosis.

B. PE – Mild cases are silent[38]. In severe PE, the patient develops dyspnoea, tachypnoea, pleuritic chest pain, cough ± haemoptysis, diaphoresis and sudden death if massive or not promptly treated.

VTE DIAGNOSIS

Deep vein thrombosis (DVT) in the symptomatic case is a clinical diagnosis made from the patient's symptoms, signs and the presence of risk factors. The previously advocated Homans sign, which is pain in the calf experienced on dorsiflexion of the foot, is now denounced. It's not only a misleading clinical sign, as it is neither sensitive nor specific for the diagnosis of DVT, but may also be associated with the risk of dislodging a clot which could precipitate a fatal PE.[13, 40]

Objective testing for DVT is however crucial because clinical assessment alone is unreliable and the consequences of misdiagnosis are serious, including severe pulmonary embolism.[41,42,43] The confirmatory investigations in the silent or suspected cases of DVT include laboratory D-dimer assay, imaging studies such as Doppler ultrasound scan (US), ascending contrast

venography, and in specialised centres, computerised tomography (CT) scan venography or magnetic resonance (MR) venography.[44]

Rather than performing the same tests in every patient, the American College of Chest Physicians (ACCP) formulated a set of guidelines in order to direct the choice of diagnostic tests for a first lower extremity DVT patient. They recommended that diagnostic test to be requested should depend on the pretest probability of VTE. In patients with low VTE risk, D-dimer or compression ultrasound (CUS) of the proximal veins of the lower limb is advocated as the diagnostic test of choice. In those with moderate risk, a highly sensitive D-dimer assay, proximal CUS, or whole leg ultrasound is advised. Patients with high pretest probability for VTE are recommended for proximal CUS, or whole leg ultrasound. [41]

However, in patients with suspected first lower extremity DVT in whom ultrasound is impractical (eg, when leg casting or excessive subcutaneous tissue or fluid prevents adequate assessment of compressibility) or non-diagnostic, the recommendation is for the patient to undergo CT scan venography or magnetic resonance (MR) venography, or MR direct thrombus imaging as an alternative to venography.

Pulmonary embolism (PE), on the other hand is confirmed with pulmonary helical computerised tomography (CT) angiography scan as the investigation of choice. Ventilation-perfusion (V/Q) scan is requested when there is a need to avoid radiation exposure or contrast from a CT scan due to a medical condition, contrast allergy or during pregnancy.[45,46] Where these facilities are not available or affordable, as in most hospitals in Africa, plain chest X-ray (CXR) may be requested. While the CXR is normal in majority of PE cases, the pathognomonic findings on CXR are the Hampton's hump (a wedge shaped radio-density seen
above the diaphragm on the affected side) and in rare cases the Westermark sign.[47,48] Chest X-ray also can demonstrate other diagnoses in patients without PE.

THROMBOPROPHYLAXIS (VTE PREVENTION)

In view of the significant morbidity and mortality associated with VTE in the surgical patient in particular, and other categories of patients in general, primary prevention strategies, otherwise termed thromboprophylaxis, have the greatest potential to cost-effectively reduce the incidence of venous thrombosis in hospitalised patients, guarantee patient safety, improve outcome and serve as a quality indicator of health care.[49, 50, 51, 52]

VTE RISK ASSESSMENT AND THROMBOPHYLAXIS MODELS

The prototypical clinical guidance tool for the identification of surgical patients at risk for VTE, assessment of their respective levels of risk and the ultimate determination of the ideal method of venous thromboprophylaxis each patient is offered is the Caprini score.[53]

On the other hand, the Padua Prediction score was formulated for the VTE assessment and prevention among medical patients.[54]

The Caprini risk assessment tool is a point-based individualised method of stratifying surgical patients. It contains several variables which include patients age, body mass index, duration of surgery > 45 minutes, type of surgery among others (Table 1). The different variables have different points ranging from 1 to 5. The Caprini score is calculated by adding the scores of all the factors present for an individual patient with the total score determining the VTE risk level.

The score is interpreted as follows:

a. Score ≤ 1 = Very low risk for VTE.
b. Score of 2 = Low risk for VTE
c. Score of 3 = Moderate risk for VTE.
d. Score of 4 = High risk for VTE.
e. Score ≥ 5 = Highest risk for VTE.

Table 1: The Caprini Score Calculator

VARIABLES	SCORE	APPLY
Age: 41 – 60 years	1	
BMI > 25kg/m^2	1	
Minor surgery	1	
Oedema of the lower extremities	1	
Varicose veins	1	
Pregnancy	1	
Post-partum	1	
Oral contraceptives	1	
Hormonal therapy	1	
Unexplained or recurrent abortion	1	
Sepsis (in the previous month)	1	
Serious lung disease such as pneumonia (in the previous month)	1	
Abnormal pulmonary function test	1	
Bed rest	1	
Inflammatory bowel disease	1	
Age: 61 – 74 years	2	
Arthroscopic surgery	2	
Laparoscopy lasting more than 45 minutes	2	
General surgery lasting more than 45 minutes	2	
Cancer	2	
Plaster cast	2	
Bed bound for more than 72 hours	2	
Central venous access	2	
Age ≥ 75 years	3	
Prior episode of VTE	3	
Positive family history of VTE	3	
Prothrombin 20210 A	3	
Factor V Leiden	3	
Lupus anticoagulants	3	
Anticardiolipin antibodies	3	
High homocysteine in blood	3	
Heparin induced thrombocytopenia (HIT)	3	
Stoke (in the previous month)	3	
Other congenital or acquired thrombophilia	5	
Fracture of the hip, pelvis or leg	5	
Elective arthroplasty	5	
Acute spinal cord injury (in the previous month)	5	
CAPRINI SCORE:		
INTERPRETATION:		

Since Caprini et al. published their initial VTE assessment and thromboprophylaxis clinical practice guidelines (CPG) in 1991 (Fig. 1), different health institutions and health regulatory authorities world wide, including the ACCP in the United States of America (USA) and National Institute for Health and Care Excellence (NICE) in the United Kingdom, have regularly issued updated adaptions of the original document to suit their peculiar practice environments and keep pace with the latest pharmacological advances in anticoagulant therapies.[9, 55] As each clinical practice guidance (CPG) differs in design and detail from others, a general protocol would be presented in this article.

AIMS AND OBJECTIVES OF CLINICAL PRACTICE GUIDELINE (CPG) FOR VTE PROPHYLAXIS

The General objective is to prevent the development of VTE in surgical inpatients and day cases during their hospital stay or within 90 days following their discharge from the hospital.[56]

The Specific objectives are to:

i. Identify all patients at risk.
ii. Determine each patient's level of VTE risk (ie risk category).
iii. Assess patient's bleeding risk.
iv. Identify and select the most appropriate prevention strategy for each risk group.

Deep Vein Thrombosis (DVT)

Prophylaxis Orders (For use in Elective General Surgery Patients)

Thrombosis Risk Factor Assessment (Choose all that apply)

BIRTH DATE

NAME

HOSP. NO

SEX M F WARD

Each Risk Factor Represents 1 Point

- Age 41-60 years
- Swollen legs (current)
- Varicose veins
- Obesity (BMI >25)
- Minor surgery planned
- Sepsis (<1 month)
- Acute myocardiai infarction
- Congestive heart failure (<1 month)
- Medical patient currently at bed rest
- History of inflammatory bowel disease
- History of prior major surgery (<1 month)
- Abnormal pulmonary function (COPD)
- Serious Lung disease including pneumonia (<1 month)
- Oral contraceptives or hormone replacement therapy
- Pregnancy or postpartum (<1 month)
- History of unexplained stillborn infant, recurrent spontaneous
- abortion (≥ 3), premature birth with toxemia or growth-restricted infant
- Other risk factors _____

Sub Total

Each Risk Factor Represents 5 Points

- Stroke (<1 month)
- Elective major lower extremity arthroplasty
- Hip, pelvis or leg fracture (<1 month)
- Acute spinal cord injury (paralysis) (<1 month)
- Q Multiple trauma (<1 month)

Sub Total

Each Risk Factor Represents 2 Points

- Age 61-74 years
- Arthroscopic surgery
- Malignancy (present or previous)
- Laparoscopic surgery (>45 minutes)
- Patient confined to bed (>72 hours)
- Immobilizing plaster cast (<1month)
- Central venous access
- Major surgery (>45 minutes)

Sub Total

Each Risk Factor Represents 3 Points

- Age 75 years or older
- History of DVT/PE
- Positive Factor V Leiden
- Elevated serum homocysteine
- Heparin-induced thrombocytopenia (HIT)

(***Do not use heparin or any low molecular weight heparin***)

- Elevated anticardiolipin antibodies
- Other congenital or acquired thrombophilia
  If yes: Type_____
  *most frequently missed risk factor
- **Family History of thrombosis**
- Positive Prothrombin 20210A
- Positive Lupus anticoagulant

Sub Total

TOTAL RISK FACTOR SCORE:

FACTORS ASSOCIATED WITH INCREASED BLEEDING

Patient may not be a candidate for anticoagulant therapy & SCDs should be considered.

Active Bleed, Ingestion of Oral Anticoagulants, Administration of glycoprotein IIb/IIIa inhibitors, History of heparin induced thrombocytopenia

CLINICAL CONSIDERATIONS FOR THE USE OF SEQUENTIAL COMPRESSION DEVICES (SCD)

Patient may not be a candidate for SCDs & alternative prophylactic measures should be considered.

Patients with Severe Peripheral Arterial Disease, CHF, Acute Superficial DVT

Total Risk Factor Score	Risk Level	Prophylaxis Regimen
0	Very Low	Early ambulation
1-2	Low	Sequential Compression Device (SCD)
3-4	Moderate	Choose **ONE** of the following medications +/- compression devices: Sequential Compression Device (SCD) - Optional Heparin 5000 units SC TDS Enoxaparin/Lovenox: 40mg SC daily (WT < 150kg, CrCI > 30mL/min) 30mg SC daily (WT < 150kg, CrCI = 10-29mL/min) 30mg SC BD (WT > 150kg, CrCI > 30mL/min) ***Please refer to Dosing Guidelines on the back of this form)***
5 or more	High	Choose **ONE** of the following medications **PLUS** compression devices: Sequential Compression Device (SCD) Heparin 5000 units SC TDS ***(Preferred with Epidurals)*** Enoxaparin/Lovenox ***(Preferred)*** 40mg SC daily (WT < 150kg, CrCI > 30mL/min) 30mg SC daily (WT < 150kg, CrCI = 10-29mL/min) 30mg SC BD (WT > 150kg, CrCI > SOmL/min) ***(Please refer to Dosing Guidelines on the back of this form)***

Ambulatory Surgery - No orders for venous thromboembolic prophylaxis required

VTE Prophylaxis Contraindicated, Reason:_____

Joseph A. Caprini, MD, MS, FACS, RV
VTE Risk Factor Assessment Tool

Physician Signature	Dr.	Date	Time
Processed by:		Date/Time	
			DVT Prophylaxis Regimen

Fig. 1: Prototypical Clinical Practice Guideline for VTE Risk Assessment and Prophylaxis Prescription.[Caprini]

THROMBOPROPHYLAXIS METHODS

There are 3 broad methods of VTE prevention in the surgical patient. They comprise the mechanical methods, pharmacological agents and interventional radiology (minimal access) procedure. These could be used either alone or in combination depending on the peculiar circumstance and level of VTE risk for each individual patient.

The general measures of ensuring adequate rehydration and early mobilisation are applicable to every surgical patient including those with no identifiable VTE risk.

SELECTING THROMBPROPHYLAXIS

As a general rule, the very low-risk surgical patients do not require specific prophylaxis other than general measures and frequent mobilisation.

Patients at low risk for VTE are managed with general measures and mechanical method using anti-embolism elastic compression stockings (ECS).

Intermediate-risk surgical patients or those with concomitant medical conditions should be considered for general measures and combined thrombophylaxis using both the ECS and low molecular weight heparin (LMWH). Intra-operatively, intermittent pneumatic calf compression (IPC) devices may be used as an alternative to antiembolism stockings, but not in addition as a means of mechanical thrombprophylaxis

All high-risk surgical patients should be similarly managed as for those with intermediate VTE risk and should in addition receive an enhanced dose of the LMWH for regimens that use daltaparin (Table 2). [7, 52, 57]

If pharmacologic prophylaxis is contraindicated, such as in patients with active bleeding or with a bleeding disorder, non-pharmacologic (mechanical) prevention should be used (Tables 2 and 3).

Antiplatelets such as acetylsalicylic acid (Aspirin) are not recommended for VTE prophylaxis in the general surgery patient.[38]

DURATION OF VTE PROPHYLAXIS

a. Thromboprophylaxis should be given throughout the period of risk which in the surgical patient may be for life

b. For general surgical procedures and medical conditions, prophylaxis can be discontinued once the patient is able to ambulate regularly and other risk factors are no longer present.

PHARMACOLOGICAL AGENTS FOR VTE PROPHYLAXIS

The choice of the medication to be used for thromboprophylaxis in the surgical patient would depend on several considerations, which include:

i. Patient's level of risk for VTE as determined from the Caprini chart.

ii. Patient's bleeding risk.

Table 2: Levels of Thromboembolism Risk and Recommended Thromboprophylaxis in Hospitalised Surgical Patients According To Patent's Characteristics.

S/No	Levels of Risk	Approximate DVT Risk Without Thromboprophylaxis (%)	Suggested Thromboprophylaxis Options
1.	Very Low Risk: Minor surgery in mobile Patients.	< 5	No specific thromboprophylaxis General measures (Adequate hydration & Early ambulation).
2.	Low Risk	<10	i. General measures ii. ECS
3.	Moderate Risk: a. Most general surgery operations.	10 - 40	General measures a. LMWH at recommended doses Or UFH bd or tds Or Fondaparinux
	b. Moderate VTE Risk + High bleeding Risk		b. Mechanical thromboprophylaxis alone
4.	High Risk: a. High VTE Risk (No high bleeding risk).	40 - 80	General measures a. LMWH at enhanced doses (if using daltaparin) Or Fundaparinux Or Vit. K Antagonist (INR 2 – 3).
	b. High VTE Risk + High Bleeding Risk		b. Mechanical thromboprophylaxis alone.

Note:

i. Patients in groups 2 to 4 with contraindications for pharmacological prophylaxis should receive mechanical prophylaxis.

ii. Major risk factors for VTE – Active cancer, History of VTE, Thrombophilia, Limb paralysis/paresis, Major trauma.

iii. Non-major risk factors for VTE – Age >40years, Anaesthesia, Obesity, Rest, Cardiac or respiratory failure, Mycardial infarctoion, Sepsis, Plane/car travel longer than 4 hours, Chemotherapy, Stroke, Family history of VTE, Central venous catheter, Hormone replacement therapy, Pregnancy, Child birth during last month.

Contraindications to pharmacological VTE-prophylaxis – Active bleeding, Platelet count < 100 X 10^9/L, Coagulopathy, Recent brain surgery, traumatic injuries with high risk for bleeding, acute haemorrhagic stroke, uncontrolled hypertension (230/120mmHg or higher), Hypersensitivity to heparin/LMWH.

Table 3. Every patient should receive LMWH

S/No.	Exception	Thromboprophylaxis
1.	Contraindications to pharmacological VTE prophylaxis	Mechanical prophylaxis
2.	Non-risk factor for VTE	Early mobilization
3.	Cancer or Previous history of VTE + surgery	LMWH + Mechanical prophylaxis
4.	Creatinine clearance < 30mls/min	Unfractionated heparin (UFH) 7500IU bd

iii. Availability of the drug.

iv. Affordability (cost) of the medication.

v. Availability of the appropriate drug monitoring investigation eg INR for warfarin.

The most extensively studied drugs for VTE prophylaxis are:

a. Unfractionated heparin (UFH).

b. Low molecular weight heparin (LMWH) eg Enoxaparin, Daltaparin and Tinzaparin.

c. Fondaparinux.

d. Warfarin – a Vitamin K antagonist (VKA).

DOSAGE

For hospitalised general surgery patients, available clinical evidence supports the prophylactic use of enoxaparin 40mg subcutaneously (SC) once daily or fondaparinux 2.5mg SC once daily for both the intermediate and high VTE risk patients. Daltaparin may be used at 2,500 units SC once daily for low and intermediate VTE risk or 5000u SC once daily for the high risk cases.[57] Tinzaparin is prescribed at a dose of 4500u SC once daily for intermediate and high risk VTE prophylaxis.

Unfractionated heparin (UFH) is given at 5000units SC 12 hourly for low and intermediate risk patients and at 7500u 12 hourly (or 5000u 8 hourly) for the patients at high VTE risk.[52]

LMWH (enoxaparin, daltaparin and tinzaparin) and fondaparinux are preferred over the UFH for VTE prevention because they are as effective, do not require monitoring at the recommended standard dose, have lower risk of heparin-induced thrombocytopaenia and they are more convenient to use at once daily dosage. However, UFH is cheaper and preferable in patients with renal impairment.[58]

The use of the newer oral anticoagulants (NOACs): rivaroxaban, dabigatran, apixaban and edoxaban for VTE prophylaxis in the general surgery patient has not been comprehensively evaluated.[59, 60, 61]

INTERVENTIONAL RADIOLOGY PROCEDURE

Inferior vena cava (IVC) Filters.

The use of IVC filter is aimed at preventing the proximal extension or embolization of DVT already present in the pelvis or lower limbs.[45,62,63] It provides long-term protection against PE in the appropriate patient. These devices are deployed in the inferior vena cava infra-renally using interventional radiology through either the femoral vein in the groin or the jugular vein in the neck.

They are indicated for patients with either recurrent DVT or those who develop DVT despite being on therapeutic doses of anticoagulation agents.

Inferior vena cava filters are also offered to general surgery patients for whom mechanical and pharmacological VTE prophylaxis are absolutely contraindicated.[56, 58]

Inferior vena cava filters are not without complications which include insertion site thrombosis, filter migration, erosion of filter through the vessel wall, recurrent DVT post-thrombotic syndrome and IVC obstruction.[64] Some authorities have therefore advocated extreme caution with their use.[65]

Due to the long-term complications of permanent IVC filters, it is recommended that a retrievable filter be used for patients with temporary contraindication to anticoagulation.[66, 67]

CONCLUSION

Venous thromboembolism (VTE) is a common but yet preventable health condition among surgical patients. The establishment of a local hospital-based clinical practice guideline (CPG) is therefore strongly recommended for the accurate VTE risk assessment, risk categorisation and also to enable the implementation of safe and effective thromboprophylaxis for every general surgery inpatient in our environment. These will significantly reduce the morbidity and mortality associated with this often unrecognised silent killer.

Conflict of interest *- Nil.*

REFERENCES

1. Shojania KG, Duncan BW, McDonald KM, et al. Making health care safer: a critical analysis of patient safety practices. Report/Technology Assessment 43, Agency for Healthcare Research and Quality, Rockville, Md, USA. 2012. Available at: http:// www.ahrq.gov/research/findings/evidence-based-reports/ ptsafetyuptp.html. Accessed March 7, 2020.

2. Venkataram A, Santhosh S, Dinakar D, Siddappa S, Babu R, Shivaswamy S. Postoperative Venous Thromboembolism Prophylaxis by General Surgeons i n a Developing Country: A Survey. Thrombosis. 2013, Article ID 873750, Available at: http://dx.doi.org/10.1155/2013/873750. Accessed March 7, 2020.

3. Lindblad B, Eriksson A, Bergqvist D. Autopsy-v e r i f i e d p u l m o n a r y e m b o l i s m i n a s u r g i c a l department: analysis of the period from 1951 to 1988. BJS 1991; 78 (7): 849–852.

4. Stein PD, Henry JW. Prevalence of acute pulmonary embolism among patients in a general hospital and at autopsy. Chest 1995; 108 (4): 978–981.

5. White RH, Zhou H, Romano PS. Incidence of s y m p t o m a t i c v e n o u s t h r o m b o e m b o l i s m a f t e r different elective or urgent surgical procedures. Thromb and Haemost. 2003; 90 (3): 446–455.

6. Beckman MG, Hooper WC, Critchley SE, Ortel TL. Venous thromboembolism: a public health concern. Am J Prev Med. 2010 (Apr; 38 (4 Suppl): S495 – 501.

7. Report of the independent expert working group on the prevention of venous thromboembolism in hospitalised patients. Department of Health. April 2007. Available at: www.dh.gov.uk/publications. Accessed March 7, 2020.

8. Geerts WH, Heit JA, Clagett GP, Pineo GF, Colwell CW, Anderson FA Jr, Wheeler HB. Prevention of venous thromboembolism. Chest. 2001 Jan; 119 (1 Suppl): 132S – 175S.

9. Geerts WH, Bergqvist D, Pineo GF, *et al.* Prevention of venous thromboembolism: American College of Chest Physicians (ACCP) Evidence-Based Clinical Practice Guidelines (8th Edition). Chest, 133 (2008), pp. 381S-4.

10. Lloyd-Jones D, Adams R, Carnethon M, *et al.* Heart disease and stroke statistics–2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation, 119 (2009), pp. e21-e181

11. Novis SJ, Havelka GE, Ostrowski D, et al. Prevention of thromboembolic events in surgical patients through the creation and implementation of a computerized risk assessment program. Journal of Vascular Surgery. 2010; 51 (3): 648-54.

12. Danwang C, Temgoua MN, Agbor VN, Tankeu AT, Noubiap JJ. Epidemiology of venous thromboembolism in Africa: a systematic review. J Thromb Haemost 2017; 15: 1770–81).

13. McCallum. P, Chetter I. Venous disorders in Bailey and Love's short practice of surgery. 26th Edition. Boca Raton London New York. CRC Press Taylor and Francis Group. 2013: 913-917.

14. Silverstein M, Heit J, Mohr D, Petterson T, O'Fallon W, Melton L. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. Arch Intern Med. 1998: 158: 585 – 593.

15. Giordano P, Grassi M, Saracco P, Molinari A, Gentilomo C, Suppiej A, etal. Paediatric venous thromboembolism: a report from the Italian registry of thrombosis in children. Blood Transfus. 2018 Jul; 16 (4): 363-370.

16.Kenet G, Nowak-Göttl U. Venous thromboembolism in neonates and children. Best Pract Res Clin Haematol. 2012;25:333–44. [PubMed] [Google Scholar]

17. Goldenberg NA, Donadini MP, Kahn SR, et al. Post-thrombotic syndrome in children: a systematic review of frequency of occurrence, validity of outcome measures, and prognostic factors. Haematologica. 2010;95:1952–9. [PMC free article] [PubMed] [Google Scholar]

18. Molinari AC, Saracco P, Cecinati V, et al. Venous thrombosis in children: an emerging issue. Blood Coagul Fibrinolysis. 2011;22:351–61. [PubMed] [Google Scholar]

19. Stein PD, Kayali F, Olson RE. Incidence of venous thromboembolism in infants and children: data from the National Hospital Discharge Survey. J Pediatr. 2004;145:563-5 [PubMed] [Google Scholar]

20. Raffini L, Huang YS, Witmer C, et al. Dramatic increase in venous thromboembolism in children's hospitals in the United States from 2001 to 2007. Pediatrics. 2009;124:1001–8. [PubMed] [Google Scholar]

21. Heit JA, Mohr DN, Silverstein MD, Petterson TM, O'Fallon WM, Melton LJ. Predictors of recurrence after deep vein thrombosis and pulmonary embolism: a population-based cohort study. Arch Intern Med. 2000; 160: 761 – 768.

22. Cushman M, Epidemiology and risk factors for venous thrombosis. Semin Haematol. 2007 Apr; 44 (2): 62 – 69.

23. Prandoni P, Lensing A, Cogo A, Cuppini S, Villalta S, Carta M, et al. The long-term clinical course of acute deep vein thrombosis. Ann Intern Med. 1996; 125: 1-7

24. Girolami A, Cosi E, Tasinato V, Santarossa C, Ferrari S, Girolami B. Drug-induced thrombophilic or prothrombotic states: an underestimated clinical problem that involves both legal and illegal compounds.

25. Ramot Y, Nyska A, Spectre G. Drug-induced thrombosis: an update. Drug. Saf. 2013 Aug; 36 (8): 585 – 603.

26. Vandenbroucke JP, Rosing J, Bloemenkamp KW, et al. Oral contraceptives and the risk of venous thrombosis. N Engl J Med. 2001;344(20):1527-1535.

27. Erman M, Abali H, Oran B, et al. Tamoxifen-induced tissue factor pathway inhibitor reduction: a clue for an a c q u i r e d t h r o m b o p h i l i c s t a t e ? A n n O n c o l . 2004;15(11):1622-1626.

28. Solak Y, Demircioglu S, Polat I, et al. Heparin-induced thrombocytopenia in a hemodialysis patient treated with fondaparinux: nephrologists between two fires. Hemodial Int. 2013;17(2): 320-323.

29. Rufa A, Cerase A, Monti L, et al. Recurrent venous t h r o m b o s i s i n c l u d i n g c e r e b r a l v e n o u s s i n u s thrombosis in a patient taking sildenafil for erectile dysfunction. J Neurol Sci. 2007;260(1-2): 293-295.

30.Talamo GP, Ibrahim S, Claxton D, Tricot GJ, Fink LM,Zangari M. Hypercoagulable states in patients with multiple myeloma can affect the thalidomide-associated venous thromboembolism. Blood Coagul Fibrinolysis. 2009; 20(5): 337-339.

31. Kleijer BC, Heerdink ER, Egberts TC, Jansen PA, van Marum RJ. Antipsychotic drug use and the risk of venous thromboembolism in elderly patients. J Clin Psychopharmacol. 2010;30(5):526-530.

32.Eekhoff EM, Rosendall FR, Vandenbroucke JP. Minor events and the risk of deep vein thrombosis. Thromb Haemost. 2000: 83: 408 – 411.

33. Cannegieter SC, Doggen CJ, van Houwelingen HC, Rosendaal FR. Travel-Related Venous Thrombosis: Results from a Large Population-Based Case Control Study (MEGA Study) PLoS Med. 2006;3 [PMC free article] [PubMed] [Google Scholar]

34. Piccioli A, Falanga A, Baccaglini U, Marchetti M, Prandoni P. Cancer and venous thromboembolism. Semin Thromb Hemost. 2006;32:694–699. [PubMed] [Google Scholar]

35.Abdollahi M, Cushman M, Rosendaal FR. Obesity: risk of venous thrombosis and the interaction with coagulation factor levels and oral contraceptive use. Thromb Haemost. 2003 Mar; 89(3): 493-8.

36.Frank B, Maher Z, Hazelton JP, et al.Venous thromboembolism after major venous injuries: Competing priorities. J Trauma Acute Care Surg. 2017 Dec;83(6):1095-1101.

37. Kennedy M, Andreescu AC, Greenblatt MS, et al. Factor V Leiden, prothrombin 20210A and the risk of venous thrombosis among cancer patients. Br J Haematol. 2005 Feb; 128(3):386-8.

38. O'Donnell M, Weitz JI. Thromboprophylaxis in surgical patients. Can J Surg. 2003; 46 (2): 129 – 135.

39. Cushman M. Inherited risk factors for venous thrombosis. Hematology Am Soc Hematol Educ Program . 2005: 452 – 457 . [PubMed] [Google Scholar]

40.Ambesh P, Obiagwu C, Shetty V. Homan's sign for deep vein thrombosis: A grain of salt? Indian Heart J. 2017 May-Jun; 69(3): 418–419.

41.Bates SM, Jaeschke R, Stevens SM, et al. Diagnosis of DVT: antithrombotic therapy and prevention of thrombosis, 9th ed. American College of Chest P h y s i c i a n s e v i d e n c e - b a s e d c l i n i c a l p r a c t i c e guidelines. 2012. Chest; 141 (2): e351–e418.

42.Bell WR , Simon TL . Current status of pulmonary thromboembolic disease: pathophysiology, diagnosis, prevention and treatment . Am Heart J . 1982 ; 103 (2): 239-262.

43. Alpert JS , Dalen JE . Epidemiology and natural history of venous thromboembolism. Prog Cardiovasc Dis . 1994 ; 36 (6): 417 - 422 .

44. Aniteye E, Wu L. Peri-operative care and post-operative complications in Baja's principles and practice of surgery including pathology in the tropics. 5th Edition (Vol. 1). Ghana. Ghana publishing corporation. 2009: 252-255.

45. Tarbox AK, Swaroop M. Pulmonary embolism. Int J Crit Illn Inj Sci. 2013 Jan-Mar; 3(1): 69–72.

46. Gottschalk A, Stein PD, Sostman HD, Matta F, Beemath A. Very low probability interpretation of V/Q lung scans in combination with low probability objective clinical assessment reliably excludes pulmonary embolism: Data from PIOPED II. J Nucl Med. 2007; 48:1411–5. [PubMed] [Google Scholar]

47. Hampton AO, Castleman B (1940). Correlation of postmortem chest teleroentgenograms with autopsy findings. Am. J Roentgenol Radium Ther. 34: 305 – 326.

48. Ray J (2003). Westermark sign and suspected pulmonary embolism. Can J Cardiol. 19 (3): 317.

49. Galante M, Languasco AN, Gotta D, et al. Venous thromboprophylaxis in general surgery ward admissions: strategies for improvement. International Journal for Quality in Health Care. 2012; Volume 24, Number 6: pp. 649 –656.

50. Clagett GP, Reisch JS. Prevention of venous thromboembolism in general surgical patients. Results of meta-analysis.AnnSurg1988,208:227-40.6

51. Anderson DR, O'Brien BJ. Cost effectiveness of the prevention and treatment of deep vein thrombosis and p u l m o n a r y e m b o l i s m . P h a r m a c o e c o n o m i c s 1997;12:17 –29.

52. Maynard G, Stein J. Preventing hospital-acquired venous thromboembolism: a guide for effective quality improvement. AHRQ Publication No. 08-0075, 2008. Agency for Healthcare Research and Q u a l i t y . A v a i l a b l e a t : http://www. ahrq . gov/qual/vtguide . Accessed 7 March, 2020.

53.Caprini JA, Arcelus JI, Hasty JH, Tamhane AC, Fabrega F (1991). Clinical assessment of venous thromboembolic risk in surgical patients. Semin Throm Hemost. 17 Suppl 3: 304-312.

54. Barbar S, Noventa F, Rossetto V, Ferrari A, Brandolin B, Perlati M et al . (2010) . A risk a s s e s s m e n t m o d e l f o r t h e i d e n t i f i c a t i o n o f hospitalized patients a r i s k f o r v e n o u s thromboembolis: the Padua Prediction Scoe. J Throm Haemost. 8 (11): 2450 – 7.

55. National Institute for Health and Clinical Excellence. Reducing the risk of venous thromboembolism (deep vein thrombosis and pulmonary embolism) in inpatients undergoing surgery. NICE clinical g u i d e l i n e N o . 4 6 : 1 – 1 6 0 . A v a i l a b l e a t : http://www.nice.org.uk/CG046. Accessed 7 march, 2020.

56. Lipp A, Loveday D, Lewis M. Norfolk and Norwich University Hospitals Trust Guideline for Prevention of Venous Thromboembolism (VTE) for Adult Surgical Patients 16 years and over. Approved by the Thrombosis and Thromboprophylaxis Committee Clinical Guidelines Assessment Panel (T&TC and CGAP). Date approved: 21/06/2019 Review date: 21/06/2022.

57. Veeramootoo D, Harrower L, Saunders R, Robinson D , C a m p b e l l W B . P r o p h y l a x i s o f v e n o u s thromboembolism in general surgery: guidelines differ and we still need local policies. Ann R Coll Surg Engl 2011; 93: 370–374

58. G u i d e l i n e f o r t h e p r e v e n t i o n o f V e n o u s Thromboembolism (VTE) in adult hospitalised patients. Published by the State of Queensland (Queensland Health), December 2018. Available at: https://www.health.qld.gov.au/clinicalpractice/guidelines-procedures/medicines/safety. Accessed March 7, 2020.

59. Thromboprophylaxis: Non-orthopaedic surgery. From Thrombosis Canada. 2016. Available at: h t t p : / / t h r o m b o s i s c a n a d a . c a / w p - content/uploads/2017/01/8.-Thromboprophylaxis-Non - Ortho - 2016Dec07 - FINAL - 1. pdf . Accessed March 7, 2020.

60. Bauersachs R, Berkowitz SD, Brenner B, Buller HR, D e c o u s u s H , G a l l u s A S , e t a l . E I N S T E I N Investigators. Oral rivaroxaban for symptomatic v e n o u s t h r o m b o e m b o l i s m . N E n g l J M e d . 2010;363:2499–510. [PubMed] [Google Scholar]

61. Büller HR, Prins MH, Lensin AW, Decousus H, Jacobson BF, Minar E, et al . EINSTEIN – PE Investigators. Oral rivaroxaban for the treatment of symptomatic pulmonary embolism. N Engl J Med. 2012;366:1287–97. [PubMed] [Google Scholar

62. Bhogal H, Eid S, Kantsiper M. When should an IVC filter be used to treat a DVT. The Hospitalist. March 2010 (3).

63. Bates SM, Ginsberg JS. Treatment of deep vein thrombosis. N Engl J Med. 2004; 351: 268 – 277.

64. Crowther MA. Inferior vena cava filters in the management of venous thromboembolism. Am J Med. 2007; 120 (10 Suppl 2): S13-17.

65. Comes RF, Mismetti P, Afshari A. European guidelines on perioperative venous thromboembolism prophylaxis: Inferior vena cava filters. European Journal of Anaesthesiology. 2018;35(2):108-11.

66. Comerota AJ. Retrievable IVC filters: a decision matrix for appropriate utilization. Perspect Vasc Surg Endovasc Ther. 2006; 18(1): 11-17.

67. Houman FM, Lopes RD, Stashenko GJ, Alexander JH, Garcia D.Treatment of venous thromboembolism: Guidelines translated for the clinician. J Thromb Thrombolysis. 2009; 28(3).